Chapter 2

Diseases of the Equine Gastrointestinal Tract

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COLIC is a nonspecific term referring to abdominal pain. In this chapter, its usage shall be restricted to gastrointestinal pain.

A. **Determining the severity** of the colic. Colic is usually sporadic in occurrence and may be mild or severe, acute or chronic. Repeated bouts in the same individual are not uncommon.

1. Patient profile and history
   a. Some intestinal problems that produce colic appear to be age related. For example, meconium impactions are restricted to neonatal foals, whereas feed impactions occur more frequently in older hones.
   b. An accurate history is essential in defining possible etiologies and pathophysiologies of medical colics. Retrospective information should include parasite-control measures, pasture size, and stocking rates. The use and work schedule for the horse should be explored, as should any changes in environment or feeding. A past and present medical history is important for diagnostic purposes as well as interpretation of presenting clinical findings.

2. Clinical findings (Table 2-1). A complete physical examination should be attempted in all cases of colic to determine the site and cause of gastrointestinal pain as well as ruling out conditions that mimic gastrointestinal pain. The examination should be performed without sedation in tractable patients. If sedation is necessary, it should be administered only after a complete general examination has been performed because sedation will affect clinically important findings.
   a. Attitude. Colics produce attitudinal changes in the horse. Mild colics (**e.g.,** large and small colon impactions) cause slight to moderate depression. Colics producing severe depression and toxemia often result from strangulation obstructions, which are not medically manageable. Medical colics may produce severe pain and anxiety, as in the case of gastric dilatation. The horse will often continue to eat with mild colics.
   b. Pain and anxiety is manifested as straining, pawing, stretching, and sweating. It is important to determine if the pain is continuous or intermittent, static or changing in intensity, responsive or unresponsive to medication.
   c. Temperature. Rectal temperature readings are usually normal to slightly elevated with medical colics. Subnormal temperatures should alert the examiner to the possibility of terminal shock and toxemia. High temperatures are associated with infectious or septic conditions. Temperatures may be normal if there has been use of antipyretic drugs (**e.g.,** dipyrone, phenylbutazone, **flunixin meglumine).**
   d. Respiratory rate. Respiratory rates usually increase in proportion to the amount of pain. Abdominal pressure creates a rapid, shallow respiratory rate and pattern. A metabolic acidosis associated with tissue devitalization causes an increase in the respiratory rate.
   e. Evaluation of circulatory status
      (1) Pulse rates reflect the nature of the colic. In the adult horse, the interpretation of the pulse rate is shown in Table 2-2.
      (2) Pulse quality should also be evaluated. A strong, full pulse (rather than a weak, thready pulse) is reflective of a mild and medically responsive colic.
      (3) Capillary refill is normal with medically responsive colics and increases with surgical colics (as a result of vascular compromise).
      (4) Normal **mucosal** color is reflective of normal circulatory status and mild or early colics. Congested mucous membranes indicate vascular compromise, fluid loss, or shock.

TABLE 2-1. Physical Findings Differentiating Mild (Medical) Colics From Severe (Surgical) Colics

Mild Colic	Severe Colic
Yawning	Rolling, thrashing, self traumatization
Straining as if to urinate	Depression, dullness
Bruxism, groaning	Labored breathing
Pawing ground	Distended abdomen
Looking at flank	Sweating
Getting up and down	Attempts at vomiting
Muscle tremors	
Possible sweating	

f. Digestive system examination

(1) Abdominal contour is usually normal with medical colics. Distention is not a feature of serious small intestinal obstruction and most commonly is observed with large intestinal problems that are usually surgical in nature.

(2) Sharp molar teeth, reflective of poor dental occlusion or improper husbandry, may predispose horses to impaction colics.

(3) Abdominal auscultation should be carried out in a comprehensive, systematic way. Normal to increased **borborygmi** usually indicate a good prognosis for medical management. **Hypermotility** indicates early intestinal distention or enteritis. Hypermotility, which results from ischemia or longstanding intestinal obstruction, may also be an initial response to gut ischemia.

(4) Method of examination

(a) During an esophageal examination, use the largest tube possible and a gentle technique, being careful to avoid esophageal perforation. To retrieve reflux, the tube may be primed with a bolus of warm water and gravity flow or suction used. The pH and composition of any fluid should be determined. Low pH fluid (4–5) indicates a gastric source, whereas a higher pH (6–7) indicates that the fluid is from the small intestine. Previously administered medications may be found in the reflux (e.g., mineral oil).

(b) A rectal examination, performed on patients of adequate size, is carried out in a systematic way, identifying normal and abnormal palpable structures. Feces may be present or absent in the rectum, a finding that is not indicative of the colic type. Firm and mucus-covered feces may point to an impaction colic. Sand in the feces is a special case of impaction colic.

(5) Abdominocentesis often is performed as part of the initial database of colic evaluation. Either a midline or paramedian site is acceptable, and the technique is considered a minor surgical procedure.

(a) A point 10–30 cm caudal to the xiphoid is chosen, and after the skin has been aseptically prepared and desensitized with a local anesthetic, either a disposable 18-gauge hypodermic needle or blunt cannula is used to penetrate the peritoneum. When using the blunt cannula, first a stab incision should be made through the skin with a scalpel blade to a depth of approximately 4 mm.

(b) The tip of a biich catheter or teat cannula is inserted through a gauze sponge, which absorbs surface blood. The catheter or cannula is then inserted through the incision.

(c) Using firm steady pressure, the instrument is advanced into the peritoneal cavity. Usually a final **"pop"** is felt when the peritoneum is penetrated.

(d) Fluid should be collected in two clear tubes, one sterile and one containing an anticoagulant, such as ethylenediaminetetraaceticacid (EDTA).

(e) Results of fluid analysis should be normal with medical colics (Tables 2-3 and 2-4).

TABLE 2-2. Interpretation of Pulse Rate*

Pulse	Rate (beats/minute)
Normal	30–39
Mild	40–59
Moderate	60–79
Serious	80–99
Severe	100+

* Foals will have relatively higher rates than adult horses.

TABLE 2-3. Laboratory Findings With Normal Equine Peritoneal Fluid

	Mean	Range
Total white blood cells ($\times 10^9$/L)	3	1–10
Neutrophils (%)	43	24–62
Lymphocytes (%)	20	4–36
Macrophages (%)	34	17–50
Eosinophils (%)	2	1–6
Red blood cells ($\times 10^{12}$/L)	0	...
Total protein (g/L)	0.1	0.05–0.15
Fibrinogen (g/L)	< 10	...
Specific gravity	1.005	1–1.015
Color	Yellow	...
Turbidity	Slightly cloudy	...

B. **Medically** manageable colics

1. **Meconium impaction**

a. Patient profile and history. Meconium passage may cause some degree of discomfort in newborn foals but is usually completed in 24–48 hours. Retention of meconium is the most common cause of colic in newborn foals.

TABLE 2-4. Gross Observation of Abnormal Peritoneal Fluid Samples

Fluid Samples	Indications
Flocculent fluid, no odor	Bacterial and toxic peritonitis seen in early infarctive disease
Serosanguineous, no odor	Leakage of RBCs, toxins, and bacteria from necrotic bowel into peritoneal cavity
Sanguinous, malodorous with fecal material	Associated with parietal pain
	Confirms presence of ruptured viscus
	Rectal tear
	Rarely, blood-tinged fluid is present
Frank blood in abdomen	Usually when blood vessel is entered or splenic parenchyma is penetrated
	Rarely neoplasms, such as hemangiosarcoma, may cause abdominal hemorrhage

RBCs = red blood cells.

b. Clinical findings. There may be repeated attempts by the foal to defecate, which is indicated by straining with an arched back, tail swishing, and restlessness. Foals may develop a high meconium impaction, which is less obvious; signs of obstruction colic and abdominal distention take longer to develop.

c. Diagnosis. Impactions usually occur in the colon and rectum and can be detected by digital examination. Radiographs are useful for demonstrating high impactions. Sonographic imaging also may be employed to evaluate bowel content, thickness, distention, and motility.

d. Treatment. Enemas (mild soap and water or commercial types) usually are adequate. Refractory cases may require repeated enemas, intravenous fluids, and finally surgical exploration.

2. Large colon **impaction**

a. Patient profile and history. This is one of the most common colic encountered in practice. Large colon impaction may be age-, feed-, or management-related and occurs with some repeatability in certain horses. Horses may have a history of dental problems, recent deworming, or feed or management changes.

b. Clinical findings. The clinical findings are consistent with a medical colic. There is often slight depression and anorexia. There are no abnormalities of temperature, pulse, and respiration (TPR), but there is evidence of periodic visceral pain when the horse stretches and looks at its flank. There is decreased fecal output, and feces are small, firm, and covered with mucus. Fecal composition may indicate the nature of the impaction (e.g., grain, sand). Rectal examination may reveal the site and the degree of the impaction. For example, the pelvic flexure is a common site of large colon impactions. On gastric intubation, there is no reflux of stomach contents. Abdominal auscultation reveals a generalized decrease in borborygmi.

c. Etiology and pathogenesis

(1) Physical agents

(a) Feed-related. Course roughage may predispose the horse to improper digestion of feedstuffs with a resultant impaction.

(b) Water-related. Insufficient amounts of water create a dry ingesta prone to impaction.

(c) Poor teeth. Similar to poorly digestible feeds, improper mastication causes some impactions.

(2) Parasitic agents. Migrating larval forms of *Strongylus vulgaris* interfere with circulation and innervation of various parts of the large intestine, which affects gut motility and leads to impactions.

(3) Extraluminal or intraluminal agents. Extraluminal events (e.g., abscesses, neoplasms, adhesions) or intraluminal masses (e.g., enteroliths) produce impaction colics. The majority of these, however, result in chronic, unresponsive colics that must be surgically managed.

d. Diagnostic plan. The clinical findings often are enough to diagnose the condition of a large intestinal obstruction. The response to therapy also is a valuable diagnostic aid.

e. Laboratory tests. Hematology and clinical pathology findings are normal. Abdominocentesis, although usually not warranted, yields fluid of normal characteristics.

f. Differential diagnoses. Differential diagnoses to consider when presented with a large intestinal obstruction include early surgical colics (e.g., strangulating obstructions, nonstrangulating small intestinal obstructions), gastric ulcers, chronic salmonellosis, chronic liver disease (cholelithiasis), and urolithiasis.

g. Therapeutic plan

(1) Analgesics. Analgesics may be indicated if discomfort levels of the horse warrant. All of the following agents may be given intravenously or intramuscularly:

(a) Flunixin meglumine: 1.1 mg/kg every 12 hours

(b) Xylazine: 0.1–1.0 mg/kg as necessary

(c) Butorphanol: 0.02–0.05 mg/kg as necessary

(d) Detornidine: 0.005–0.03 mg/kg as necessary

(e) Pentazocine: 0.3 mg/kg as necessary

(2) Laxatives

(a) Laxatives and wetting agents aid in softening the mass. The following substances are all oral medications:

(i) Mineral oil: 2–4 L every 12 hours

(ii) Dioctyl sodium sulfosuccinate (DSS): 10–20 mg/kg

(iii) Bran mashes

(b) Intravenous or oral fluids also may be employed to soften intestinal masses. Doses are empirical.

(3) Surgery may be necessary if the condition persists, worsens, or if clinical signs become repetitive.

h. Prevention

(1) Revisits to the patient or a client's attention to clinical signs are necessary to judge the response to therapy. If repeated doses of analgesics are necessary to control the pain or if the pain increases in intensity or duration, the diagnosis of a primary medically responsive large intestinal impaction must be reassessed. A decision for surgery must be made early for the maximum probability of success.

(2) Clients need to consider management changes in order to address the risk factors (e.g., feed types, feeding techniques, access to water, dental management, proper parasite control).

3. Distention colic (spasmodic or gas colic)

a. Patient profile and history. This is a commonly diagnosed colic with similar subjective findings to other medical colics. Horses that crib (windsuck) often seem predisposed to distention colic.

b. Clinical findings. As with other medical colics, there might be slight increases in TPR. On abdominal auscultation, there may be increased peristaltic activity, particularly between bouts of pain. Abdominal percussion may reveal tympanic sounds of intestinal gas. There will be minimal reflux on nasogastric intubation. Often during a rectal examination, bowel distended with gas is felt.

c. Etiology and pathogenesis

(1) Simple distention colics result from intestinal spasm or ileus. The intestine distends with fluid and gas cranial to the site(s) of spasm, causing visceral pain. Peristalsis may increase in the distended segments due to local myoelectrical stimulation.

(2) The initial cause of the intestinal spasm or ileus may be related to the same risk factors associated with the development of simple obstruction colics (i.e., parasite migration, feed changes, management deficiencies).

(3) As a special case of distention colics, horses that crib and swallow air cause gastric distention and pain.

d. Diagnostic plan. The clinical findings should be compared with the degree of pain and response to therapy. Simple distention colics may appear similar to early cases of obstruction colics, strangulating obstruction colics, and nonstrangulating infarctions, which are more serious and may require surgical intervention.

e. Laboratory tests. Laboratory values are not outside of normal ranges for this condition.

f. Differential diagnoses. The following categories of colics and specific conditions are surgical in nature but early in their course may appear similar to a simple intestinal distention.

(1) **Nonstrangulating obstructions**

(a) Foreign bodies

(b) Ascarid impactions (young animals)

(c) Meconium impaction (foals)

(d) Muscular hypertrophy of the ileum

(e) Pedunculated lipomas

(f) Abscesses

(g) Adhesions

(h) Neoplasms

(2) Strangulating obstructions

(a) Small intestinal volvulus

(b) Strangulating lipomas
(c) Intestinal entrapment—epiploic foramen entrapment, omental defects, mesenteric defects, nephrosplenic ligament entrapment, hernias (inguinal, umbilical, scrotal, diaphragmatic)

(3) **Nonstrangulating infarctions**

g. **Therapeutic plan.** Treat with analgesics, laxatives, and wetting agents similar to a large colon impaction.

h. **Prevention.** Discuss treatment, management, and prevention in a similar way to other medical colics.

4. **Proximal enteritis (anterior enteritis, duodenitis)** is an idiopathic syndrome characterized by ileus and transluminal leakage of fluid into the gut. Horses with this condition exhibit moderate colic but marked depression. There are diminished gut sounds and copious gastric reflux.

a. **Patient profile and history.** This disorder is seen primarily in adult horses. Proximal enteritis is not common in occurrence but is similar in appearance to a small intestinal obstruction, which presents as a surgical colic. There may be a history of recent grain engorgement or heavy grain feeding.

b. **Clinical findings.** This colic is usually mild, but affected horses are extremely depressed.
(1) A **fever** is often evident (38.5°C–40.0°C), and the heart rate is increased (60–120 beats/min). An elevated respiratory rate is caused by pain.
(2) The horse may be **dehydrated,** with resultant signs of fluid volume depletion (e.g., dry and injected mucus membranes, increased capillary refill time, decreased skin elasticity).
(3) **Gastric reflux** is invariably present with several liters being retrievable. The reflux is green-yellow with an alkaline pH (6–7) indicating small intestinal origin.
(4) **Peristaltic** sounds are weak, and rectal palpation reveals a slightly distended small intestine. This distention does not increase over time. The course of the disease is 7–10 days.

c. **Etiology and pathogenesis**
(1) **Etiology.** Suggested causes include pancreatitis, clostridiosis, verminous arteritis, and gram-negative enteritis (e.g., salmonellosis).
(2) **Pathogenesis.** The gastric reflux produces the pain associated with the condition. There is an associated **toxemia** with varying signs of shock, coagulopathy, laminitis, and renal dysfunction. **Postmortem findings** demonstrate inflammation and degeneration of duodenal intestinal mucosa. A fibrinopurulent exudate is present on the serosal surface. Lesions are less commonly found in the jejunum or at the pylorus.

d. **Diagnostic plan.** The determination of a clinical diagnosis is challenging. A horse with mild to moderate pain, severe depression, and fever is more likely to have an enteritis than small intestinal obstruction. There is, however, gastric reflux in both instances. A small intestinal obstruction causes a progressive deterioration in clinical signs, whereas a proximal enteritis has a steadier course and pain that is relieved by nasogastric intubation.

e. **Laboratory tests**
(1) The packed cell volume (PCV) and total serum protein (TSP) are elevated, which indicates **dehydration.**
(2) There is **hypokalemia** due to potassium sequestration in the small intestine.
(3) There may be a **hypochloremic, metabolic alkalosis** due to HCl pooling in the stomach, but serum electrolytes are usually within normal ranges.
(4) The **complete blood cell count (CBC)** may show a white blood cell picture indicative of an infective or inflammatory response.
(5) Results of an **abdominocentesis** are nondiagnostic. There may be an increase in protein content of the abdominal fluid, but cellular components are usually normal.

f. **Differential diagnosis.** Rule out small intestinal colics of a surgical nature (e.g., strangulating obstructions). Also, consider primary causes of enterocolitis (e.g., salmonellosis).

g. **Therapeutic plan.** The therapy remains empirical but needs to be aggressive. Removal of gastric fluid must be carried out by repeated or continuous nasogastric intubation. Large volumes of intravenous fluids (balance electrolytes or saline, depending on blood pH) should be administered. Pharmacologic agents include flunixin meglumine (0.25 mg/kg twice daily) and penicillin–gentamicin combination. It may also be necessary to treat concurrent disease (e.g., laminitis; see Chapter 13 III A 5). Therapy may need to be continued for up to 10 days before gastric reflux ceases and normal eating resumes.

5. **Flatulent colic (tympany, bloat, wind colic)**

a. **Patient profile and history.** Adult horses are affected with this type of colic under conditions of recent stress or management changes. Histories might include any of the following:
(1) Overfeeding on highly fermentable feed
(2) Cold-water engorgement
(3) Feeding while exhausted or overheated
(d) Moldy hay or grain feed
(5) Behavioral abnormalities (e.g., cribbing, greedy eaters)
(6) Medication administration, such as atropine or broad spectrum antibiotics

b. **Clinical findings**
(1) Animals appear in **distress** and TPR is elevated out of proportion to other clinical signs.
(2) **Abdominal distention** may be evident if gas is contained in the colon or cecum. Cecal tympany causes filling in the right paralumbar fossa, whereas bilateral abdominal distention occurs with large colon gas accumulation.
(3) **Gastric distention** is not evident externally. Simultaneous auscultation and percussion may reveal the location of the distended viscus.
(d) **Pain** and signs of colic accompany the visceral distention. Often, the **distended gut** is palpable per the rectum (excluding cranial viscera, such as the stomach). Passage of a nasogastric tube often relieves any gastric distention.

c. **Etiology and pathogenesis**
(1) Flatulent or tympanic colic results from **excessive gas accumulation** in the intestinal tract. The overdistention of viscera stimulates pain and pressure receptors, causing mild to severe colics. The severe forms mimic surgical colics.
(2) **Gaseous distention** usually is caused by increased fermentation and gas production, air accumulation (e.g., as with cribbing), or ineffectual gastrointestinal tract motility, causing gas buildup.
(3) Gas may accumulate anywhere along the gastrointestinal tract, resulting in some variation in the clinical findings.

d. **Diagnostic plan.** This condition is diagnosed on clinical findings. It is important to note the disproportionate degree of pain relative to other findings that are absent (e.g., shock). This helps differentiate the condition as a medical colic.

e. **Laboratory tests.** Laboratory findings are normal on hematology and abdominocentesis.

f. **Therapeutic plan**
(1) Employ **nasogastric intubation** to relieve stomach distention if this is the source of the colic. Treat with sedatives and analgesics (e.g., xylazine, butorphanol, pentazocine, detomidine). Administer mineral oil for its anti-fermentative properties.
(2) If medical therapy is ineffective, **trocarization** of the cecum or colon may provide relief. This is a relatively radical therapy but may provide temporary relief until medical intervention can work to alleviate the problem. Trocarization must be carried out in an aseptic manner.
(a) Determine the site of trocarization by simultaneous auscultation and percussion.
(b) Block the site with local anesthetic, then use a number 15 scalpel blade to pierce the skin.
(c) A 14-cm trocar is used to penetrate the abdominal wall and lumen of the bowel. Hold the trocar in place until gas is no longer free flowing.

(d) Inject 10–20 ml of antibiotic as the trocar is withdrawn, and place the hone on systemic antibiotics.

g. Prevention. The prognosis for uncomplicated cases of flatulent colic is usually favorable.

II. DIARRHEA.

The large intestine of the horse has tremendous water absorptive capabilities. Diarrhea (acute or chronic) results from large intestinal pathology or pathological changes to the small intestine that cause an overwhelming amount of fluid and ingesta to be presented to the large intestine.

A. Acute diarrhea in adult horses

1. **Salmonellosis**

a. Patient profile and history. Salmonellosis is usually a sporadic disease in single animals'unless in a referral center setting or in a barn or stable with frequent animal movement on and off premises. Salmonellosis infection may take various forms. The acute diarrheic form is most often seen in weanlings and young performance horses that are stressed following transport, shows, or surgery.

b. Clinical findings. Clinical salmonellosis has a spectrum of clinical expression. The enteric form in the adult may be asymptomatic, mild, acute severe, or chronic.

(1) Mild infections are associated with fever, anorexia, depression, and the production of poorly formed feces (cow-pie).

(2) With acute severe infections, fever and depression is seen during the first 24-48 hours. Simultaneous with this is mild to severe abdominal pain. At this time, the condition can be confused with a surgical colic. Diarrhea begins sometime after the initial signs but may take 2–4 days to develop. Diarrhea is projectile, foul smelling, and persistent. Expression of diarrhea often is accompanied by improvement in the other clinical signs. Horses usually continue to eat, but in the case of anorexic animals, the prognosis for survival is poor.

(3) The diarrhea may persist for 3–4 weeks, at which time horses will have experienced significant weight loss. Ventral edema caused by hypoproteinemia also may be a finding. Laminitis is a frequent sequela to salmonellosis.

(4) A peracute form of enteric salmonellosis may occur; affected horses die within 6–12 hours.

c. Etiology and pathogenesis

(1) Etiology. Saimoneiia *typhimurium* is the isolate most commonly associated with equine diarrhea (60% of cases). The organism adheres to and invades the mucosa of the intestine. The development of enteritis is then dependent on factors such as the age of host, immune status, other stressors, and virulence of the organism strain.

(2) Pathogenesis

(a) Diarrhea and enteritis result from the effects of the bacteria and host inflammatory mediators (prostaglandins).There is an increased secretion of chloride, sodium, and water into the intestinal lumen via an increase in mucosal cell cyclic adenosine nmnophosphate (cAMP) content.

(b) The characteristic fever and leukopenia are caused by the release of lipopolysaccharide endotoxin from the bacterial cell wall. White blood cells pool at the site of the infection, and protein leakage occurs across permeable intestinal vessel walls.

d. Diagnostic plan. The diagnosis is based on clinical findings supported by laboratory confirmation.

e. Laboratory tests

(1) Hematologic findings are a neutropenia with a left shift and varying degrees of cellular morphologic changes (toxicity). The albumin fraction of the TSP is low, although the total protein may be elevated or normal due to dehydration; The PCV is elevated due to dehydration, and the horse will have a metabolic acidosis with electrolyte losses through the feces.

(2) Fecal culture for Salmonella may be unrewarding because of the dilution effect of diarrhea and the adherent nature of the bacteria to the intestinal mucosa. A rectal mucosal biopsy may enhance the likelihood of culturing the organism.

f. Differential diagnoses include equine monocytic ehrlichiosis, intestinal clostridiosis, antibiotic-induced diarrhea, proximal enteritis, and small intestinal obstruction.

g. Therapeutic plan

(1) Of primary consideration is fluid replacement therapy, with large volumes of alkalinizing fluids.

(2) The use of antibiotics is controversial and perhaps best restricted to bacteremic or septicemic manifestations of salmonellosis in foals. If antibiotics are used in the enteric form of the disease, those with gram-negative specificity are recommended (e.g., gentamicin, amikacin, trimethoprim–sulfa combinations). Flunixin meglumine is recommended for its anti-inflammatory effect.

(3) Bismuth subsalicylate is recommended as an intestinal protectant and antiprostaglandin. It turns the feces black.

(4) Plasma transfusions may be necessary in hypoproteinemic horses. Heparin may be used in cases of coagulopathies [e.g., disseminated intravascular coagulation (DIC)] associated with the disease.

h. Prevention

(1) Horses with diarrhea should be isolated in a separate barn if possible. Caretakers should wear dedicated and protective clothing. A foot bath should be used at the entrance to the facility. Manure from cases should be handled and disposed of in a secure and separate way. Salmonella species are zoonotic.

(2) In-contact animals should be cultured to identify fecal shedders. External sources of contamination (e.g., feed) should be examined particularly if multiple serotypes appear in several animals. Monitor stablemates for evidence of increases in body temperature or the appearance of lassitude. A total white blood cell count may be performed on suspect animals, and if a neutropenia is present (which often precedes clinical signs), isolation and therapy should be instituted.

(3) Most common disinfectants are effective against Salmonella species; however, sanitation is challenging because of the difficulty of removing all organic material. Recovered horses may become shedders, some chronically. However, shedding for more than 6 weeks is uncommon with most serotypes.

2. Equine monocytic ehrlichiosis (acute equine diarrhea syndrome, Potomac horse fever)

a. Patient profile and history

(1) First described in the United States Northeast, this disease is now evident throughout North America and has been recorded in Europe. It is seasonal in occurrence, with summer being the most common time of incidence. Any age group of animal may be affected, but the disease peaks in adult animals at age 12 years. It is most often found in Thoroughbred horses on pasture. Females are more at risk than males, and it is usually sporadic with single horses on any given farm.

(2) The owner may report a mild depression and anorexia followed by diarrhea.

b. Clinical findings

(1) Cardinal signs are anorexia, fever (39.5°C), injected mucous membranes, and depression. A profuse, watery diarrhea commences 24–48 hours after the onset of fever and lasts up to 10 days in the majority of animals. Mild abdominal pain with decreased borborygmi is evident.

(2) Laminitis may be a sequela in 25% of cases. Occasional horses may show injected mucous membranes, severe abdominal distention, and abdominal pain. Frequently, death ensues before diarrhea develops. Abortion may occur in pregnant mares.

c. Etiology and pathogenesis. *Ehrlichia risticii* is the etiologic agent. The organism has a predilection for mononuclear cells and is hypothesized to be spread by an arthropod vector.
d. Diagnostic plan. Indirect fluorescent antibody (FA) may be performed on serum collected at 1- to 3-week intervals. Serum should be separated promptly and submitted cool but not frozen because freezing lowers the antibody titre. A latex agglutination test has also been developed for diagnosis.
e. Therapeutic plan. Supportive care is essential, as with any acute enteritis. Tetracycline at 6.6 mg/kg administered intravenously once per day (if given 24 hours after the onset of fever) for 5 days results in a dramatic response. Diarrhea does not develop. Treatment with tetracyclines after the onset of diarrhea does not alter the course of the disease.
f. Prevention. Treatment is costly and often futile when full clinical signs develop. A vaccine, is now available that Seems to protect approximately 75% of horses.

3. **Colitis** X
a. Patient profile and history. This is a sporadic disease associated with a history of recent stress in adult horses.
b. Clinical findings. A short febrile period is followed by a normal to subnormal body temperature. There is marked tachypnea, hyperpnea, and depression. There is a rapidly developing, intense dehydration and occasional abdominal pain. The hone may die before diarrhea is evident.
c. Etiology and pathogenesis. The causative agent is believed to be *Clostridium perfringens* type A. Clinical signs result from an enterotoxemia.
d. Diagnostic plan. The condition usually is diagnosed post mortem.
e. Therapeutic plan. Intensive therapy with massive quantities of isotonic saline and added bicarbonate is required to combat the dehydration and metabolic acidosis. Supplemental potassium therapy also may be necessary. Plasma transfusions may be warranted if hypoproteinemia is present. Flunixin meglumine and heparin also may be employed. Antimicrobial therapy may include penicillin–aminoglycoside or trimethoprim–sulfa combinations.
f. Prevention. Little can be recommended to the client to prevent or treat this highly fatal, sporadic disease.

4. Antibiotic-associatedenteritis
a. Patient profile and history. There are anecdotal reports of enteritis in horses following antibiotic administration. Tetracycline is the antibiotic most often incriminated, but lincomycin, tylosin, and high doses of penicillin and erythromycin also have been associated with the disease. There also have been reports of diarrhea after the use of trimethoprim–sulfadrugs.
b. Clinical findings. The frequency of enteritis associated with most of these drugs is low enough that they continue to be used when indicated. Typical signs of acute enteritis develop. Signs may subside rapidly when the antibiotics are discontinued.
c. Etiology and pathogenesis Antibiotics may upset the normal gut flora, allowing overgrowth by nonpathogenic or pathogenic bacteria. Pathogenic bacteria (e.g., salmonella, clostridia), when established, may have rapidly fatal consequences. Occasionally, chronic diarrhea has been seen in association with Salmonella isolated from the feces.
d. Therapeutic plan. Discontinue the antibiotic use, and treat as other acute diarrheas.

5. **Intestinal clostridiosis**
a. Patient profile and **history.** The disease affects horses that are most commonly over 1 year of age. The disease is sporadic and may be accompanied by the history of recent, severe stress. Although reported as a distinct condition, intestinal clostridiosis may be similar or identical to colitis X.
b. Clinical findings. The disease is of peracute onset with profound depression, tachycardia, dehydration, and diarrhea, which is profuse and malodorous. Shock is evidenced by a rapid heart rate and cardiovascular compromise. Affected animals die within 24 hours of the onset of clinical signs.

c. Etiology and pathogenesis. C. perfringens type A is the etiologic agent.
d. Laboratory tests. C. perfringens counts may be performed on the feces. Laboratory findings are consistent with dehydration and circulatory collapse.
e. Therapeutic plan. Massive fluid therapy is essential for any hope of success. Antibiotics are of little value, but penicillins may be employed as a logical choice for antibacterial therapy.

6. Gastrointestinal ulceration [nonsteroidal anti-inflammatory drug (NSAID) toxicity]
a. Patient profile and history. Foals and young animals are extremely susceptible to NSAIDs. However, older horses occasionally are affected if the manufacturer's recommended dosages are grossly exceeded.
b. Clinical findings. Diarrhea is an occasional clinical finding, but more commonly the condition is associated with recurrent abdominal pain, anorexia, and weight loss. Oral ulceration with excessive salivation may be evident. Dependent edema may be a finding.
c. Etiology and pathogenesis. NSAIDs (e.g., phenylbutazone) produce toxic side effects if used in excess or in dehydrated horses. Organ systems most commonly affected include the gastrointestinal tract, kidneys, and hematopoietic system. Toxicity of the gastrointestinal tract results from depletion of protective prostaglandins (such as PE_2). These prostaglandins normally decrease gastric acid secretion and increase the protective layer of gastric mucosa. The agents also may produce vasoconstriction, resulting in devitalization and ulceration of mucosa along the entire intestinal tract. Oral ulceration is caused by the local irritative effect of the drug.
d. Diagnostic plan. Diagnosis is based most often on clinical findings and a history of long-standing or overzealous use of phenylbutazone. The diagnosis may be supported by endoscopy of the stomach or double-contrast gastric radiography in foals and ponies. These techniques reveal ulceration of the glandular portion of the stomach.
e. Laboratory tests. Laboratory tests may be helpful by revealing a hypoproteinemia and hypoalbuminemia from protein leakage across a reduced and devitalized gastrointestinal mucosa. Occult blood may be found in the feces, accompanied by a lowered hernatocrit.
f. Therapeutic plan
(1) Discontinue all NSAIDs, and administer 1–2 g/100 kg of sucralfate orally four times daily and 6 mg/kg cimetidine orally, intravenously, or intramuscularly 2–3 times per day. Ranitidine may be substituted for cimetidine at 1–3 mg/kg orally twice daily or at 0.5 mg/kg intravenously twice daily. Plasma transfusions may be warranted in cases of severe hypoproteinemia.
(2) Intravenous feeding or nasogastric intubation and alimentation may be necessary.
g. **Prevention.** Phenylbutazone should be used with caution in ponies, younger horses, and dehydrated animals. Foals that are heavily parasitized or malnourished are extremely prone to toxic side effects.

7. **Fungal** enteritis
a. Patient profile and history. Fungal enteritides are sporadic in occurrence.
b. Clinical findings. The condition is indistinguishable from other acute diarrheas. Cases usually present with severe toxemia, profound dehydration, and severe, profuse, watery diarrhea.
c. Etiology and pathogenesis. Fungal overgrowth of the gastrointestinal tract and lungs may occur due to immunocompromise or secondary to extensive antibiotic use.
d. Diagnostic plan. There is little help for diagnosis. Fecal fungal elements occasionally may be found.
e. Therapeutic plan. There is no known treatment

B. Diarrhea in nursing foals

1. Foal heat diarrhea

a. Patient profile and history. Foals often develop diarrhea between 6 and 14 days of age. This may correspond with the dam's first postpartum estrus.
b. Clinical findings. The foal presents with soft to watery feces, but all other signs are usually within normal limits. There may be mild dehydration, but foals are generally alert with normal appetites. The condition is most often self-limiting in 2–3 days but may precede other diarrheas in the same foal.
c. Etiology and pathogenesis. The etiology of foal heat diarrhea is unknown but may be associated with a changeover of cell type as the intestinal mucosa of the neonate matures. Other postulated but less likely causes include hormonal or nutritional alterations in the mare's milk, coprophagia, Strongyloides westeri infestation, and alterations in intestinal microbiological flora.
d. Diagnostic plan. The diagnosis usually is based on clinical findings without the need for laboratory suppott.
e. Therapeutic plan. Therapy is dictated by the severity of the diarrhea. Uncomplicated cases may be treated with simple attention to nursing care, such as washing the perineum and applying petroleum jelly. If diarrhea persists beyond 3 days, treatment with *1–2* ml/kg bismuth subsalicylate four times per day orally and oral fluid replacement with commercial calf formulations should be considered.

2. Nutritional diarrheas'
a. Clinical findings. Diarrhea may range from soft feces to very watery stool. Other clinical findings are usually normal, and the foal may have a normal attitude and appetite.
b. Etiology and pathogenesis. Diarrhea may develop secondarily to the following situations:
(1) Ingestion of excessive amounts of milk. This may occur with foals that are greedy eaters or when the mare and foal are reunited after a period of separation. Normally, a milk clot forms in the stomach within minutes of ingestion, and the whey advances to the small intestine in gradual amounts as the clot contracts. Overingestion can result in excessive amounts of whey entering the duodenum, overwhelming absorptive capabilities and creating an osmotic drive towards fluid accumulation in the gut.
(2) Abnormal nursing. Foals that ingest milk too rapidly or are fed by nasogastric tube experience decreased salivary secretion, which adversely affects milk dilution and clot digestion.
(3) Sudden dietary changes
(4) Ingestion of fibrous material. Grain, forage, mare's feces, or other fibrous material require digestion in the immature large intestine of the foal. This promotes indigestion and diarrhea.
(5) Carbohydrate intolerance. Young foals may have primary or secondary carbohydrate intolerance. Primary milk intolerance is relatively rare. Secondary carbohydrate intolerance results from an enteric infection, which causes an increase in mucosal cell turnover. More immature cells make up the absorptive cell component of the gut mucosa, decreasing the disaccharidase and absorptive activities in the mucosal brush border.
c. Diagnostic plan. The diagnosis is most often based on clinical findings and history. Diarrhea is usually self-limiting but may be unresponsive in the case of primary carbohydrate intolerance.
d. Laboratory tests. Laboratory findings are unrewarding. In the case of an unresponsive milk intolerance, a lactose tolerance test may be performed. Lactose is administered per os, and corresponding blood glucose levels are determined.
e. Therapeutic plan
(1) Nursing care may be the only therapy necessary in the case of short-lived diarrheas. Lactose intolerance presents a special case, in that continued exposure to a milk diet exacerbates the problem.
(2) Elimination of milk and dietary replacement with hand feeding of a commercial soy-based milk supplement may be necessary until the foal can be weaned back on whole milk. A commercial calf diarrhea oral replacement solution may aid in the recovery.

(3) A commercially available lactase enzyme preparation may be added to milk before feeding to partially digest lactose into its constituent monosaccharides.

3. Antibiotic-related diarrheas
a. Patient profile and history. Oral antibiotics, such as aminoglycosides, may kill normal gut flora and predispose foals to diarrhea. Systemic antibiotics with an enterohepatic circulation (oxytetracycline, lincomycin, erythromycin) also have been shown to induce diarrhea.
b. Clinical findings. Diarrhea may range from soft feces to very watery stool. The foal's appearance may range from systemically normal to significantly dehydrated with circulatory collapse.
c. Diagnostic plan. The diagnosis is based on clinical signs, history, and response to therapy.
d. Therapeutic plan. Stop antibiotic therapy. Oral administration of a slurry of fresh feces from an older horse may be beneficial in restoration of gut flora but is not proven for efficacy.

4. Mechanical irritation
a. Patient profile and history. Young or older foals may be affected by consuming inordinate amounts of sand or dirt. Sand has an abrasive effect on the intestinal mucosa, resulting in enteritis and diarrhea. Also, physical impaction may result from the accumulation of sediment.
b. Diagnostic plan. Demonstration of sand in the feces aids in the diagnosis. Sand can be seen by mixing feces with water in a rectal sleeve, then identifying and quantifying the gritty sediment.
c. Therapeutic plan. Repeated therapy is necessary. Mineral oil may be used if an impaction is suspected. However, for elimination of sand over time, an agent producing fecal bulk is preferable. Bran, in the case of older foals, or psyllium hydrophylia muciloid is recommended at 0.5 kg/454 kg four times per day orally for days or weeks.
d. Prevention. Foals or horses that actively eat soil are difficult to manage. Feeding from elevated mangers and ensuring adequate pasture cover may be effective in preventing further cases.

5. Diarrhea caused by *Strongyloides westeri*
a. Patient profile and history
(1) S. westeri infestations occur when the foal ingests infective larvae in the dam's milk. The greatest number of larvae are shed 2–3 weeks postpartum. The prepatent period is *8–14* days.
(2) It is speculated that diarrhea is associated with larval burdens. The larvae may cause intestinal mucosal damage and suppression of disaccharidase or polypeptidase production. However, foals with high fecal egg counts may have no evidence of diarrhea. Conversely, diarrhea may occur in foals with very low fecal egg counts. Thus, causation is speculative.
b. Diagnostic plan. The diagnosis may be strengthened by the presence of *S.* westeri larvae in the mare's milk or the characteristic embryonated eggs in the foal's feces.
c. Therapeutic plan. Various anthelmintics are effective against the adult parasites, including ivermectin at 200 μg/kg, thiabendazole at 50 mg/kg, cambendazole at 20 mg/kg, and oxibendazole at 10 mg/kg. The daily administration of cambendazole at 30 mg/kg to postparturient mares eliminates infective larvae in the milk for the duration of therapy.

6. Bacterial enteric disease
a. Patient profile and history. The incidence of bacterial-induced diarrheas in foals is much lower than in other domestic species. Generally, if bacteria cause the diarrhea, there is a concomitant systemic disease. These systemic conditions (e.g., salmonellosis, *Actinobacillus equuli* bacteremia) are covered in Chapter 18.
b. Etiology and pathogenesis
(1) Enteric colibacillosis caused by Escherichia coli has been documented but is

of very low incidence. Normal foal management practices may limit the occurrence of this condition.

(2) *Clostridium* perfringens types A, B, and C have been associated with enteritis and death in foals. *C. difficile* may be involved in the pathogenesis of acute enterocolitis or a mote mild diarrhea.

c. Differential diagnoses. Rule out causes of septicemia.

d. Therapeutic plan. Treatment of bacterial enteric disease is as for neonatal calves [see Chapter 3 II B 3 a (6)].

7. Viral enteritis

a. Patient profile and history

(1) Rotavirus is a definitive cause of diarrhea in foals. Clinical signs occur in foals under 3 months of age. Diarrhea can occur in individual animals or in farm outbreaks. Rotavirus in foals produces a profuse watery diarrhea, and foals may become dehydrated, depressed, and anorexic. The diarrhea may last for days or weeks.

(2) Coronavirus has been associated with diarrhea, but an etiologic relationship has not been established.

(3) Adenovirus produces diarrhea as part of a severe clinical disease of combined immunodeficiency (CID) in Arabian foals.

(4) Chronically affected foals become unthrifty as mucosal cell damage results in nutrient malabsorption.

b. Etiology and pathogenesis. Enteric viruses produce gut pathology. Rotavirus is a prime example of gut pathology. The virus invades enteric mucosal cells, causing absorptive cell loss and compensatory crypt-cell hyperplasia with a resultant malabsorptive and hypersecretory diarrhea.

c. Diagnostic plan. The virus is shed in greatest quantity early in the infection. Diagnosis depends on the demonstration of virus in the feces through electronmicroscopy, enzyme-linked immunosorbent assay (ELISA), or latex agglutination.

d. Therapeutic plan. Treatment consists of nursing care and oral or intravenous fluids. Bismuth subsalicylate and lactobacillus culture may be administered orally. Total parenteral nutrition may be indicated if diarrhea is persistent.

e. Prevention. Rotavirus persists in the environment and may be intermittently shed by carriers. The disease may occur under conditions of poor hygiene, overcrowding, or stress. Vaccination has not proven successful. Bovine colostrum, containing rotavirus antibodies, has provided some protection.

8. **Cryptosporidiosis**

a. Patient profile and history. The disease occurs in foals less often than in calves but has been documented as a cause of diarrhea in foals 5 days to 6 weeks of age.

b. **Clinical** findings. The diarrhea is of varying severity and self-limiting. *Cryptosporidium* species often act in concert with other agents, which complicates the clinical picture. Dehydration, anorexia, and weight loss all may accompany the diarrhea.

c. Etiology and pathogenesis. The protozoan parasite, *C. parvum*, is ingested as a sporulated oocyst and matures through six major developmental stages. Some maturation events occur within the cells of the distal small intestine, cecum, and colon. Villous atrophy with malabsorption and diarrhea result.

d. Diagnostic plan. The disease must be differentiated from other causes of neonatal diarrhea in foals.

e. Laboratory tests. Fecal oocysts can be detected by the staining of fecal smears, fecal flotation, or immunofluorescence.

f. Therapeutic plan. There is no known treatment for cryptosporidiosis, and animals are cared for symptomatically, with oral or intravenous fluids if necessary.

g. Prevention. The transmission of infective oocysts is through a fecal–oral route. Therefore, hygiene and management changes may be warranted if a number of foals are affected. The disease is a zoonosis and is of particular concern in immunocompromised people. Proper personal hygiene is imperative when handling infective cases.

C. Diarrhea in weanlings and yearlings

1. Parasite burdens

a. Patient profile and history. Intestinal parasites produce diarrhea and other clinical signs relative to the parasite. Intestinal parasites are ubiquitous and, under conditions of poor management techniques, produce serious problems.

b. Clinical findings. The clinical findings with intestinal parasite infestation include diarrhea, poor weight gain, unthrifty appearance, colic, depression, inappetence, and occasional elevations in body temperature.

c. Etiology and pathogenesis

(1) Parascaris equorum has a prepatent period of 10–13 weeks and will cause diarrhea if present in large numbers. Foals ingest eggs from the environment, and migrating larvae induce inflammatory lesions in various viscera. The infection is very common in foals, but a natural immunity soon develops. Eggs are shed by infected animals and are highly resistant to the elements.

(2) *Strongylus vulgaris* and *S. edentatus* induce diarrhea primarily by larval migration. The prepatent periods are 6 months and 11 months, respectively for the two parasites. Foals are infected by ingesting infective eggs while grazing contaminated pastures. *S. vulgaris* more commonly causes diarrhea.

d. Diagnostic plan. Diagnosis depends on clinical findings, laboratory tests, and response to therapy.

e. Laboratory tests. Fecal egg flotation aids in the diagnosis, although negative flotations in the face of significant worm burdens may be expected before the shedding of eggs by adult worms. Conversely, the presence of strongyle eggs in the feces of young foals may be caused by coprophagia.

f. Therapeutic plan

(1) For *P. equorum*, piperazine at 88 mg of base/kg is effective for the removal of adult worms. Foals should be dewormed at 8-week intervals to remove the adult worms before patency.

(2) Several of the benzimidazoles, as well as pyrantel pamoate and ivermectin, are effective against *S. vulgaris* larvae.

g. Prevention. An effective parasite-control program cannot be overemphasized. This should include deworming often and at regular intervals, pasture decontamination, and sound hygiene practices.

2. **Salmonellosis**

a. Patient profile and history. Salmonella species, most commonly *S. typhimurium*, produce an acute diarrhea or septicemia in any age horse.

b. Diagnosis and recommendations for therapy and control are the same as for the adult (see II A 1).

3. Rhodococcus *equi* diarrhea

a. Patient profile and history. **Affected** foals range between 4–8 months of age, and there is usually a history of respiratory infection or R. equi isolation on the farm.

b. **Clinical** findings. Feces have a variable consistency but are usually watery. Weight loss is common.

c. Etiology and pathogenesis

(1) The organism may be ubiquitous in the environment with many foals exposed; however, comparatively few foals develop clinical disease. Route of infection by R. equi is aerogenous [see Chapter 7 I A 2 b (2)]. Bacteria also may gain access via the oral route.

(2) Parasitic larval migration may spread the bacteria through the lungs or colon, seeding down viscera with abscesses. Gastrointestinal disease may occur without primary lung involvement

(3) Gastrointestinal signs are referable to abscesses within the gastrointestinal wall or mesenteric lymph nodes.

d. Diagnostic plan. A history of R. equi pneumonia on the premise aids in diagnosis, but a definitive diagnosis is difficult.

e. Laboratory tests. Fecal staining and culture may reveal R. equi; however, the organism may be shed in the feces of normal foals. There may be an elevation in γ-globulins and fibrinogen, as well as a leukocytosis.

f. Therapeutic plan. Extended therapy with erythromycin in combination with rifampin is recommended for the respiratory disease [see Chapter 7 I A 2 b (2) (f)].
g. Prevention. The prognosis is poor to grave with intestinal disease caused by R. *equi*.

III. WEIGHT LOSS

A. Granulomatous enteritis (lymphocytic-plasmacytic enteritis)

1. Patient profile and history. This condition is relatively uncommon, sporadic in occurrence, and found in adult horses. There is no demonstrable breed or sex predilection. The history usually includes weight loss despite adequate nutrition and feed consumption.
2. Clinical findings. Diarrhea is infrequent, but thinness of the animal is evident. The horse may have ventral edema. Rectal examination may reveal a roughened, friable rectal mucosa, thick-walled intestines, and prominent mesenteric lymph nodes or abdominal masses.
3. Etiology and pathogenesis
   a. This condition appears to be an immunologic phenomenon that is poorly understood. It may present as a spectrum of inflammatory bowel disease in the horse. The bowel disease results in a malabsorption syndrome.
   b. Agents postulated to be involved in this immune-mediated condition include dietary constituents, cell wall components, infectious agents (e.g., mycobacteria), and internal parasites (e.g., *Strongylus vulgaris* larvae, cyathostomes).
   c. Granulomatous changes are most evident in the mucosa of the small intestine. Histological examination reveals infiltration of the lamina propria by mononuclear cells. Absorptive capabilities are compromised and protein leakage occurs into the lumen.
4. Diagnostic plan. The diagnosis is based on history, clinical findings, laboratory data, and the unresponsiveness of the condition.
5. Laboratory tests. There is marked hypoproteinemia and hypoalbuminemia. Occasionally, acid-fast organisms may be found in the feces or on rectal biopsy. Specific carbohydrate absorption tests (e.g., D-xylose absorption test) may show reduced absorption capabilities. Concurrent albumin loss may be measured by labeled albumin clearance into the feces (chromium 51).
6. Therapeutic plan. Therapy is not affective but is often attempted before a definitive diagnosis. Treatments include nutritional support, antiparasitasides, and corticosteroids.

B. Chronic eosinophilic gastroenteritis. This disease is similar in most respects to granulomatous enteritis and the resultant malabsorptive process. Differences include:

1. Incidence in younger horses
2. The excessive infiltration of eosinophils that is found throughout the gastrointestinal tract, associated organs, and skin

C. Lymphosarcoma

1. Patient profile and history. There is a wide age range of affected horses, but younger horses are more commonly affected.
2. Clinical findings. The clinical findings depend on the organ system involved, but if the gastrointestinal system is infiltrated by lymphocytes, the condition resembles other infiltrative diseases. If the skin or superficial lymph nodes are involved, the most obvious clinical finding may be cutaneous nodules.

3. Etiology and pathogenesis. No causative agent has been associated with this tumor in horses.
4. Diagnostic plan. The diagnosis is based on clinical findings, laboratory data, and the unresponsiveness of the condition.
5. Laboratory tests. Neoplastic lymphocytes may be found in a peripheral blood smear, A bone marrow aspirate or abdominocentesis may show neoplastic lymphocytes. There will be hypoproteinemia and hypoalbuminemia. Liver enzymes may be elevated with hepatic involvement.
6. Therapeutic plan. There is no treatment for this condition.

D. Gastric ulceration/gastritis (see II A 6)

E. Parasitism

1. Patient profile and history. Parasitism is one of the most common causes of weight loss and chronic diarrhea in horses. It should not be overlooked, particularly in younger animals. Parasitism is most clinically significant in the young, weak, or stressed animal. Management factors, such as overcrowding, inadequate nutrition, and neglecting pasture rotation, or parasite prophylaxis all greatly impact the internal parasite burdens. It is most commonly a disease of populations of horses. Poor thrift and recurrent abdominal pain (episodes of colic) may be historical findings in individual animals.
2. Clinical findings. The animal may present as thin with a poor haircoat. Diarrhea of varying degrees is a common finding. If a rectal examination can be performed, the anterior mesenteric artery may feel roughened or exhibit fremitus. Inappetence, anemia, and a low-grade fever also may be present to varying degrees.
3. Etiology and pathogenesis
   a. Large strongyles cause intestinal ischemia through the migration of larval forms within the walls of blood vessels supplying portions of the large intestine. Intestinal damage may also be caused directly, as larvae mature within the walls of the large intestine and cecum and emerge into the lumen. Ulceration and erosion of the cecum and colon also result from feeding of adult strongyles.
   b. Cyathostomes (small strongyles) cause less damage, except under the specific conditions of simultaneous maturation of many hypobiotic larvae. In this case, significant intestinal damage occurs with resultant diarrhea and rapid weight loss.
4. Diagnostic plan. The diagnosis is most often made on clinical signs and environmental and management history. Other hones on the premises often show evidence of harboring a parasite burden. For individual animals where verminous arteritis is considered, ultrasonography may be an aid to diagnosis.
5. Laboratory tests
   a. Fecal egg counts are the best diagnostic but may be negative if clinical signs are caused primarily by larval forms of the parasite(s). Egg counts are also affected by host immunity, species of parasite, and history of treatment.
   b. Clinical pathology findings of some help in individually affected animals include eosinophilia, an increase in β-globulins, and an abdominocentesis consistent with chronic abscessation (macrophages with ingested bacteria, eosinophilia, increased protein content, increased leukocytes).
6. Therapeutic plan. There are many effective, broad spectrum anthelmintics that may be used for large strongyle or cyathostome infestation.
   a. The most common and efficacious treatments are:
      (1) Ivermectin paste—0.2 mg/kg
      (2) Oxibendazole—10 mg/kg
      (3) Benzimidazoles plus piperazine
   b. For migrating larval forms of the strongyles producing verminous arteritis, the following treatments may be employed:
      (1) Ivermectin at 0.2 mg/kg and oxfendazole at 10 mg/kg in single doses

(2) Fenbendazole—7.5–10 mg/kg daily for 5 days
(3) Thiabendazole—440 mg/kg daily for 2 successive days

7. Prevention. Prevention of heavy parasite loads revolve around recommendations that address pasture rotation, manure removal, and routine, effective deworming programs.

F. Abdominal abscess

1. Patient profile and history. There are few subjective findings of diagnostic note. History and management factors may indicate parasitism as a possibility with secondary abscessation of vascular lesions.
2. Clinical findings. Clinical findings are ill-thrift, fever of undetermined origin, and digestive disturbances (e.g., chronic abdominal pain, diarrhea, impaction colic).
3. Etiology and pathogenesis. Agents such as Streptococcus *equi* and *S. zooepidemicus* may be seeded down in verminous tracts, invade the abdomen through hematogenous spread, or extend into the abdomen from the gastrointestinal tract. Abscesses and abdominal adhesions may affect bowel motility and produce signs of ill-thrift, diarrhea, fever, and inappetence.
4. Diagnostic plan. Clinical findings are nonspecific.
5. laboratory tests. Laboratory findings may be helpful by revealing a leukocytosis, neutrophilia, and hyperfibrinogenemia. Protein electrophoresis may show an increased globulin fraction. Abdominocentesis exhibits an increase in leukocytes, protein, and bacteria. Culture of abdominal fluid is warranted to determine a pathogen.
6. Therapeutic plan. Long-term antibiotic therapy is indicated. Surgical exploration may be warranted in select cases.

STUDY QUESTIONS

DIRECTIONS: Each of the numbered items or incomplete statements in this section is followed by answers or by completions of the statement. Select the ONE numbered answer or completion that is BEST in each case.

1. What is the most common cause of colic in newborn foals?

(1) Meconium impaction
(2) Atresia ani
(3) Atresia coli
(4) Ascarid impaction
(5) Gastric ulceration

2. Which one of the following statements regarding distention colics (also known as spasmodic colics) is true?

(1) They are accompanied by very high pulse rates.
(2) They are not at all similar in presentation to obstruction colics.
(3) They produce large quantities of gastric reflux.
(4) They cause reflex intestinal atony.
(5) They are seen with greater frequency in horses that swallow air.

3. Which statement regarding proximal enteritis is true? Proximal enteritis:

(1) causes signs of severe colic in affected nones.
(2) may be seen in horses with a recent history of grain engorgement.
(3) is best treated by observation.
(4) causes sequestration of large amounts of fluid in the large intestine.
(5) is seen primarily in juvenile horses (yearlings).

4. In horses, the diarrheic form of salmonellosis and equine monocytic ehrlichiosis differ in what way?

(1) Fever and depression is exhibited with equine monocytic ehrlichiosis but not with salmonellosis.
(2) Fluid replacement therapy is not necessary with equine monocytic ehrlichiosis.
(3) Laminitis is a frequent sequela to salmonellosis but not to equine monocytic ehrlichiosis.
(4) Salmonella organisms invade the intestinal mucosa, whereas *Ehrlichia risticii* invade mononuclear cells.
(5) *Salmonella* are readily isolated from the feces of horses with salmonellosis, whereas *Ehrlichia risticii* cannot be recovered.

5. Which statement regarding viral diarrheas in foals is true? Viral diarrheas in foals:

(1) do not produce changes to enteric cell morphology.
(2) can be prevented by vaccination.
(3) have been associated with infection by rotavirus, coronavirus, and adenovirus.
(4) are acute but very short-lived.
(5) are diagnosed by analysis of paired serum samples.

6. Abdominocentesis is often performed in horses with colic. Which one of the following statements is correct?

(1) Peritoneal fluid with normal cell counts and low protein levels will be retrieved in cases of early, pelvic flexure impactions.
(2) The collection of peritoneal fluid contaminated with ingesta confirms the presence of a ruptured viscus.
(3) The retrieval of frank blood on abdominocentesis indicates gastric ulceration.
(4) Peritoneal fluid can be obtained from most normal horses but should have a low total white blood cell (WBC) count and high protein level.
(5) Malodorous peritoneal fluid confirms the presence of acute diffuse nonseptic peritonitis.

7. Which one of the following sets of clinical signs would be compatible with a medically manageable equine impaction colic?

(1) A pulse of *56* beatsimin, a capillary refill time of *5* seconds, and a rectal temperature of *36.7" C*
(2) A capillary refill time of 2 seconds, a respiratory rate of 16 breathsimin, and a negative retrieval of fluids on gastric intubation
(3) Peritoneal surfaces that feel granular on rectal palpation, a respiratory rate of *36* breathsimin, and a negative retrieval of fluid on gastric intubation
(4) Increased borborygmi on abdominal auscultation, a capillary refill time of 5 seconds, and warm feet with an easily palpable digital pulse
(5) Anorexia, a rigid, splinted abdomen, and a pulse of 76 beats/min

DIRECTIONS: Each of the numbered items or incomplete statements in this section is negatively phrased, as indicated by a capitalized word such as NOT, LEAST, or EXCEPT. Select the ONE numbered answer or completion that is BEST in each case.

8. All of the following statements concerning proximal enteritis of horses are true EXCEPT:

(1) colic signs may be mild to moderate but the patient is often depressed and dehydrated.
(2) repeated or continuous gastric decompression is therapeutic.
(3) it is a condition usually found in foals or weanlings.
(4) this condition is very similar in presentation to a strangulating small intestinal obstruction.
(5) laminitis may be a complication of proximal enteritis.

9. Which one of the following agents is NOT thought to cause acute diarrhea in adult horses?

(1) Enterotoxigenic Escherichia coli
(2) *Ehrlichia risticii*
(3) Clostridium perfringens type A
(4) Salmonella *typhimurium*
(5) Clostridium *difficile*

10. All of the following statements comparing large intestinal impaction colics to flatulent colics in horses are true EXCEPT:

(1) both colic types may be feed related.
(2) both colic types may be relieved by treatment with mineral oil.
(3) clinical pathology findings are usually normal with both colic types.
(4) both colic types are seen more commonly in mature horses.
(5) both colic types are usually severely painful.

ANSWERS AND EXPLANATIONS

1. The answer is **1** [I B 1 a]. Meconium impaction is much more common than the congenital atresias in foals. Ascarid impaction and gastric ulceration may occur relatively frequently but in older foals.

2. The answer is **5** [I B 3 a]. Air swallowing results in pain due to gastric or small intestinal distention. This is not the only or most common cause of distention colic, but it will produce colic signs more frequently in horses with this vice. Distention colics do not result in major changes to vital signs or cause fluid reflux to accumulate in the stomach. Distention of intestinal segments usually result in a reflex hyperperistalsis in adjacent portions of the bowel. Distention colics may appear very similar to early obstruction colics or other more serious colics in early stages.

3. The answer is 2 [I B 4 a]. Grain engorgement or heavy grain feeding in adult horses is associated with the development of proximal enteritis. Colic signs are minimal with this condition but depression is significant. Treatment must be aggressive and consist of gastric decompression, large volumes of intravenous fluids, analgesics, and antibiotics.

4. The answer is **4** [II A 1, 2]. These organisms have predilection for different tissues even though resultant clinical signs may be similar. Both diseases cause horses to exhibit fever, depression, and often a subsequent laminitis. Both require aggressive fluid replacement therapy. Salmonella species often cannot be easily isolated from the feces of affected horses because of the dilution nature of the clinical diarrhea and the invasive nature of the organism.

5. The answer is **3** [II B 7]. Rotavirus causes diarrhea in foals, whereas coronavirus has been demonstrated in the feces of diarrheic foals and adenovirus involved with the diarrhea seen in foals with combined immunodeficiency. Viral diarrheas may be short or protracted in duration and have not proven to be preventable through vaccination. Diagnosis is usually via examination of the feces.

6. The answer is 1 [I A 2 f (5) (e); Table 2-3; Table 2-4]. Peritoneal fluid findings on abdominocentesis will be normal (i.e., normal cell counts, low protein levels) in patients with early pelvic flexure impactions (medical colic). Ingesta on abdominocentesis may indicate rupture of a viscus or penetration of the intestine by the needle or cannula. Gastric ulcers may bleed, but blood would be confined to the lumen of the gastrointestinal tract. Frank blood often indicates penetration of the spleen during the procedure. Malodorous peritoneal fluid on retrieval would suggest a septic process.

7. The answer is **1** [I A 2, B 2]. A pulse of 56 beats/min, a capillary refill time of 5 seconds, and a rectal temperature of 36.7°C would be the clinical findings most consistent with equine impaction colic, which is most commonly treated medically. The condition presents with normal vital signs and little reflux on nasogastric intubation. The impaction may be palpable per rectum. Generally, increased respiratory rates and capillary refill times are associated with surgical colics.

8. The answer is 3 [I B 4]. Proximal enteritis (anterior enteritis, duodenitis) is most common in adult hones, not foals or weanlings. The condition may be confused with a strangulating small intestinal obstruction. Continuous or periodic gastric decompression relieves the colicky signs and depression becomes the major finding. Dehydration is a reflection of gastric pooling of fluid and decreased fluid intake. Laminitis, a common complication of proximal enteritis, is believed to occur secondary to an endotoxemic state.

9. The answer is 1 [II A, B 6 b (1)]. Enterotoxigenic Escherichia coli causes neonatal diarrhea in many species and has been isolated from the feces of foals with diarrhea; however, its clinical significance is unproven in foals and it has not been shown to be cause of acute diarrhea in adult horses. Escherichia risticii, Clostridium perfringens type A, Salmonella typhimurium, and Clostridium *difficile* have been associated with diarrhea in adult horses.

10. The answer is 5 [I B 2, 5]. Large intestinal impactions are only mildly to moderately painful, whereas flatulent colics are very painful and may appear to be surgical in nature. Both colic types may be feed-related—an impaction of the large intestine may be related to improper digestion of feedstuffs, whereas a flatulent colic is often associated with grazing on succulent green feeds. Mineral oil can be beneficial in the treatment of both types of colics (i.e., it may coat an impaction, allowing for easier passage and it may act as an antifermentative in the case of flatulent colics). Clinical pathology findings are usually normal for both conditions and both conditions are most common in adult horses.